TĤE ATES PATENT AND TRADEMARK OFFICE

Attorney Docket No.:

Group Art Unit:

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Examiner:

Unknown

For:

Serial No.: 07/946

METHOD OF TREATING TNF-DEPENDENT INFLAMMATION USING TUMOR NECROSIS

**FACTOR ANTAGONISTS** 

INFORMATION DISCLOSURE STA

Hon. Commissioner of Patents and Trademarks Washington, D.C. 20231

Dear Sir:

duplicate Pursuant to the provisions of 37 CFR 1.56, 1.97 and 1.98, applicants hereby provide copies of documents listed on the attached Form PTO-1449.

Documents AAA, AAB, AAC, AAD, AAE, D, G, and H were cited in the International Search Report of applicants' corresponding PCT application.

Document AAA reports the cloning of a cDNA of a p80 human TNF receptor. Also reported are the sequence similarities of the cloned protein to nerve growth factor and a transcriptionally active open reading frame of Shope fibroma virus, thereby defining a family.

Document AAB reports the cDNA cloning of a p60 and a p80 form of TNF receptor, and the identification of one of the urinary TNF-binding proteins.

Documents AAC and AAD report the cloning and expression of the human p60 TNF receptor protein and the high degree of sequence homology to the nerve growth factor receptor extracellular domain.

Document AAE reports the isolation from human urine of TNF binding proteins I and II. The isolation was performed on a TNF affinity column. Similarity in structure and immunological cross-reactivity between TNFBP-I and II and the cell surface TNF receptors is suggested.

Document D reports the identification of a 33 kDa protein which exhibits TNF-α inhibitory activity. The protein was purified from human urine and a short N-terminal amino acid sequence is disclosed.

Documents G reports the purification of TNF binding protein-II. The document also reports the generation of antibodies against the TNF-binding protein-II.

Document H reports the preparation of monoclonal antibodies against a cell surface TNF binding protein. No disclosure is provided of a TNFR amino acid sequence or DNA sequence.

Documents B, AW, AX, AH, and AAF were cited in the International Preliminary Examination of applicants' corresponding PCT application. Document B reports a process for determining the sensitivity of cells to the effect of TNF by measuring the number of TNF receptors on the cells. Monoclonal antibodies against the TNF receptors are reported.

Document AW reports reports the clones of the IL-1 receptor and its characterization as a member of the immunoglobulin superfamily. A cDNA sequence and the deduced amino acid sequence of the murine IL-1 receptor and its characterization as a member of the immunoglobulin superfamily.

Document AX reports the purification of a TNF receptor using highly purified TNF. No disclosure is made of an amino acid sequence or a cDNA sequence of the receptor.

Document AH reports the purification by immunoaffinity chromatography of the p60 TNF receptor. Other characteristics of the p60 TNF receptor are provided, but no sequences of the receptor protein or cDNA are provided.

Document AAF reports a soluble CD4 molecule which binds gp120. TNF receptors are not disclosed.

Documents A, C, D, E, F, G, AA, AB, AC, AD, AE, AF, AG, AH, AI, AJ, AK, AL, AM, AN, AO, AP, AQ, AR, AS, AT, AU, AV, AW, AY and AZ are of record in the parent application, serial number 07/523,635 and are described therein.

Since the submission of the above documents is made before the mailing of the first Office Action in connection with the above-captioned application, no fee under 37 CFR 1.17(p) is believed to be required. However, should a fee be necessary, applicants authorize the Commissioner to charge deposit account No. 09-0089 in an amount necessary to permit consideration of this Information Disclosure Statement.

Respectfully submitted

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## **CERTIFICATE OF MAILING**

I hereby certify that this correspondence is being deposited with the United States Postal Service as first class mail in an envelope addressed to: Commissioner of Patents and Trademarks, Washington, D.C. 20231, on the date indicated below.

april 21, 1993 Signed: Kimberly E. Perter